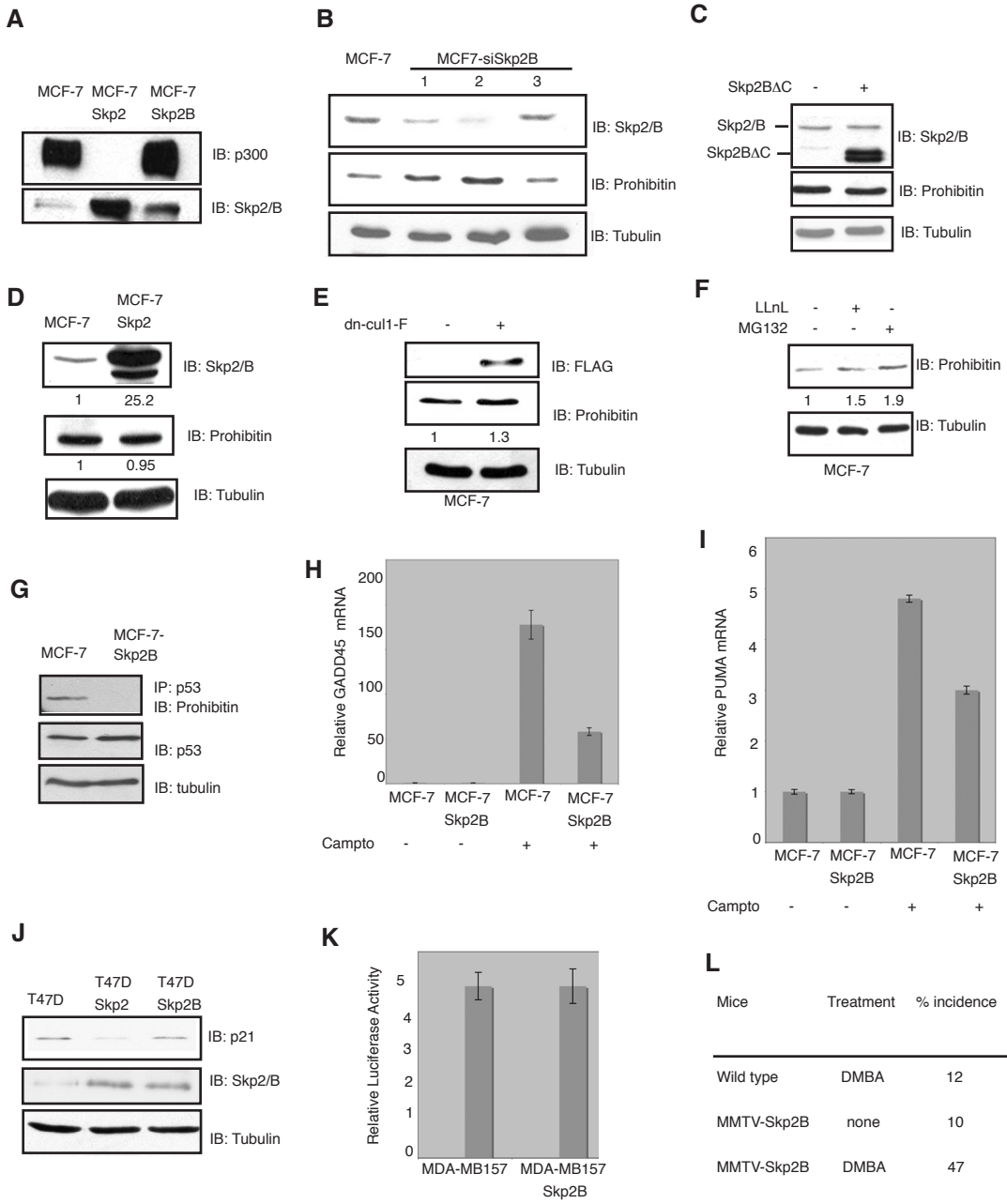
**Suppl-Fig 1**

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| Mice | Treatment | % incidence | Latency (weeks) |
|---|---|---|---|
| Wild type | DMBA | 12 | 17 |
| MMTV-Skp2B | none | 10 | >52 |
| MMTV-Skp2B | DMBA | 47 | 17 |

Legend to supplementary material

A) MCF-7, MCF-7-Skp2 and MCF-7 Skp2B cells were analyzed by Western blotting for p300 levels.

B) MCF-7 cells were transfected with three different siRNA against Skp2B and the levels of prohibtin tested by Western blotting.

C) MCF-7 cells were transfected with 5μg of Skp2BΔC plasmid and 48 hours later cell were harvested for western analysis of the levels of Skp2BΔCand prohibitin. Tubulin was used as a loading control.

D) The levels of Skp2 and prohibitin was determined by Western blot in MCF-7 cells and MCF-7-Skp2 stable clones.

E) MCF-7 cells were transiently transfected with dominant-negative cul-1 plasmid (5 μg). Cells were harvested 24 hours later for Western analysis of prohibitin and Flag-Cul1.

F) MCF-7 cells were treated with 50 μM LLnL and 20 μM MG132 for 24 hours. Cells were harvested for Western analysis of prohibitin.

G) Immunoprecipitation (IP) of endogenous p53 and prohibitin in MCF-7 and MCF-7-Skp2B cells. Following immunoprecipitation, prohibitin was analyzed by Western blot . The levels of p53 were also determined and tubulin was used as a loading control.

H) MCF-7 and MCF-7Skp2B cells were treated with 10 μM camptothecin for 12 hours and cells were harvested to determine the levels of endogenous GADD45 mRNA using 100ng total RNA.

I) MCF-7 and MCF-7Skp2B cells were treated with 10 μM camptothecin for 12 hours and cells were harvested to determine the levels of endogenous GADD45 mRNA using 100ng total RNA.

J) T47D, T47D-Skp2 and T47D-Skp2B stable clones were used form Western analysis of p21. Tubulin was used as a loading control.

K) The p53 null breast cancer cells MDA-MB 157 cells were transfected with 5 μg Skp2B plasmid and 1μg of p21 promoter luciferase reporter, 24 hours later the cells were treated with 10μM camptothecin for 24 hours and the activity of the p53 reporter measured.

L) Table of summary of the percentage incidence of mammary tumor and their latency in wild type mice treated with DMBA (7,12-dimethyl-1, 2 benzanthrace) (Sigma), MMTV-Skp2B transgenic mice treated with DMBA or no treatment. For DMBA treatment, DMBA was dissolved in corn oil at a concentration of 10μg/ml. Seven weeks old virgin female transgenic and wild type mice were treated with 4 weekly doses of 100 $\mu$l of a 10μg/ml solution of DMBA by gavage. The mice were checked weekly for mammary tumor formation by palpation. After 17 weeks, the mammary glands of mice were harvested for histological analysis.